Carotid Artery Noninvasive Testing is Being Overused

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SUMMARY The most frequent tests done in the evaluation of patients with potential cerebrovascular disease are Oculoplethysmography (OPG) and Imaging procedures. These procedures are useful in the evaluation of asymptomatic carotid bruits and may be helpful in evaluating patients with nonhemispheric symptoms. However, there is no reasonable indication for the use of these noninvasive procedures on patients with lateralizing hemispheric or ocular transient ischemic attacks. Lateralizing transient ischemic attacks are very often caused by ulcerated non-stenosing lesions which are easily missed by these noninvasive means. Arteriography is mandatory in this group whenever the patient is a potential surgical candidate. In the event of lateralizing nonhemispheric transient ischemic attacks (TIA's) are not due to the restriction of blood flow imposed by a stenosis but rather to microembolization from an ulcerated plaque or some protrusion or recess in the flow path. If an ulceration is not associated with a severe stenosis, it will be missed by OPG. Therefore in TIA's, when the patient is fit for an operation, an arteriogram is mandatory whether the OPG is positive (stenosis, occlusion) or negative (possibility of ulceration). Given that the necessary arteriogram provides more and better information than an OPG, why are OPG's done at all in patients with TIA's? Low-suction OPG methods (Kartchner, Zira) could supposedly avoid arteriograms in that small number of patients who have a bruit over the bifurcation secondary to an external carotid lesion but have a normal internal carotid artery. However, because of the notorious unreliability of the ear lobe traces one could only forego arteriography after a scanner-obtained image shows the normal internal and stenosed external carotid arteries.

OPG-Gee is a good method of obtaining retinal artery pressures. It has proven useful in anticipating the contribution of the other vessels to the flow of a hemisphere whose carotid supply is to be cut off, usually because of malignant disease in the neck. I find the OPG-Gee a good tool to determine and document the contribution of a revascularization procedure to hemispheric (eye) pressures. This is relevant information in the design and study of new surgical techniques.

OPG methods are also used in the workup of patients with non-hemispheric ischemic brain symptoms. Patients with these symptoms are often referred to vascular laboratories before completing a medical workup to rule out the far more frequent medical causes of vertebrobasilar insufficiency such as orthostatism, medication, or cardiac problems. The OPG is done in these patients to see if a stenosing carotid lesion might explain the symptoms. The trouble is that in patients without lateralizing symptoms only a minority will have extracranial carotid or vertebral disease as the cause of their symptoms. Those with severe carotid
stenoses will be identified but the rare patients with pure vertebral artery disease will be missed because of negative OPG’s, and, indeed, normal carotid arteries. It is probably sensible to use OPG as a screening test in these patients to uncover a silent and severe internal carotid stenosis but only after the more common and non-arterial causes of vertebrobasilar insufficiency have been investigated. In the event of a normal OPG, the possibility of pure vertebral disease can only be ruled out by angiography.

Oculoplethysmography is, without a doubt, useful in the followup of carotid endarterectomies, where it can pick up lesions such as restenosis secondary to intimal hyperplasia. Also, and particularly in conjunction with an imaging procedure, it can reassure us that an asymptomatic neck bruit is of external rather than internal carotid origin.

**Imaging**

More complex noninvasive techniques such as *Pulsed Doppler Ultrasound* and *B-mode scanning* give an image of the carotid bifurcation. Unfortunately, ultrasound beams cannot pierce calcium. This results in acoustical shadows (B-mode) or no signal at all (pulsed Doppler) whenever calcium is encountered either in the bones framing the cervical carotid artery (jaw and clavicle) or in the arterial wall. Calcium in the wall may give an image similar to a stenosis. Sampling of normal velocity waveforms distal to a sonopaque plaque unmasks the fictitious stenosis caused by a calcium shadow but an ulceration within such shadow cannot be detected.

When good selective angiography is available, there is no test which can better outline a carotid lesion. The arteriogram, of course, also provides information about the intracranial vessels and the branches of the arch often essential to the choice of treatment.

**Bruit Analysis**

The clinical correlation between the pitch of a bruit and the tightness of the underlying arterial stenosis has been pursued by technology. A simple amplifier-recording system such as the one designed by Kartchner and McCrae is helpful though not indispensable in localizing noises and determining their propagation.

Equipment is being sold which analyzes noises and estimates residual lumen diameter with a microcomputer. Accuracies of ± 1 mm in 93% of cases, or slightly less, are reported. While this sounds precise it means that the lumen estimated to be 2 mm in diameter may actually have 1.0 mm to 3.0 mm diameter. This wide range encompasses most of the lesions bound to produce noise anyhow.

Leaving aside other questions on the assumptions behind these computations, how is our workup or treatment to change if the residual lumen is 1.5 mm versus 2.7 mm? The decision to do an angiogram or to operate cannot be determined by these values. The clinically important features of a stenosis (location, ulceration, surface thrombus) cannot be defined by bruit analysis.

On the other hand, the very tight (98% or so) stenotic lesions, those one might like to know about in a hurry, are precisely the ones likely to be silent and thus go undetected. One must then conclude that the estimation of lumen diameter within ± 1 mm is a datum of little relevance in making a clinical decision.

In summary, a few treatment decisions can be made by using noninvasive methods in the management of patients with potential cerebrovascular disease. The main use of these methods in clinical practice is the identification of a bruit as being of an external or internal carotid source and the detection of postoperative carotid artery abnormalities, mostly restenosis. Screening for occult severe carotid lesions before an operation involving blood loss or hypotension can be done with the OPG although the translation of a positive finding into a new treatment plan is still in question. In patients with lateralizing TIA’s noninvasive methods do not add anything but delay to the information given by a good quality 4-vessel arteriogram which is mandatory if the patient is fit for an operation. In patients with non-lateralizing TIA’s the more common medical causes should be investigated beforehand. In this group the noninvasive tests will be conclusive only when they are positive and prompt an arteriogram.

The clinical and commercial interest in these noninvasive techniques has produced sophisticated equipment capable of giving us valuable information until now lacking: data on the natural history of a lesion and its modifications by various treatments. The combination of B-mode ultrasound and pulsed gated Doppler may provide important information on the rate of growth and possibly the regression of carotid lesions now that these outlines can be obtained with good optical resolution. This basic information is needed but, for the time being, it is research information and not for day-to-day clinical management.

It is pertinent to remember that the considerations just made presume good technique and understanding of the basic pathology and anatomy involved. Unfortunately, some of these noninvasive tests are done by a person whose basic experience was acquired in a 2 or 3-week familiarization course given by the manufacturer of the equipment. The test may then be read by a physician who only remotely supervised it. Under these conditions the limited but undeniable usefulness of these tests becomes a liability since false negative and false positive reporting will increase.

From my perspective as a surgeon, what is happening to noninvasive techniques has a certain similarity to that which followed the advent of electromagnetic flow meters. We thought then that they provided an essential measure to any arterial reconstruction. We quoted Lord Kelvin’s aphorism often and recorded faithfully flow rate data and waveforms before and after arterial reconstructions. Even a new noun — flowmetry — was coined. We learned about waveforms and flow rates under various conditions but in general we failed to show that the addition of a single flow datum could indicate an operation or predict its
outcome. The market interest in flow meters, however, resulted in reliable machines which are today invaluable in our experimental laboratories.

I anticipate a similar outcome for much of the noninvasive instrumentation. It will be used in larger centers where patients are referred for difficult diagnostic problems and where it is necessary to document the hemodynamic effects of new procedures or study specific changes in accessible arteries. Disillusion with some of the claims made and improvements in computer-enhanced intravenous arteriography — in itself, not a panacea — will give noninvasive tools a smaller but well deserved place in larger medical centers but outside of day-to-day clinical practice where I believe they are used in excess.

References
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Comments

Carotid Artery Noninvasive Testing Is Being Overused

ROBERT S. LEES, M.D.

To the Editor:
The paper by Berguer represents a particular school of thought concerning noninvasive carotid testing which is considerably out of date. The author’s point that the validity of noninvasive test results depends upon good technique as well as an understanding of the basic pathology and anatomy involved is a good one and an important one. His implicit advice that noninvasive laboratories be closely supervised by physicians is equally sound. Beyond those two points, however, I find little with which to agree with, and many gaps in Dr. Berguer’s knowledge.

First and foremost, the natural history of carotid disease is not “research information . . . not for day-to-day clinical management.” It is most critical for management of patients. There is for instance, no proof that ulcerated carotid lesions embolize, except rarely, in the absence of significant stenosis. In many patients in whom a TIA or stroke is attributed to such a lesion, the source of embolus is in reality the aortic arch, a left ventricular mural thrombus from an undetected earlier myocardial infarction, the left atrium from undetected rheumatic heart disease, or the mitral valve itself, with anappreciated mitral prolapse, to name a few relatively common proximal sites of thromboembolism. The angiogram is perhaps a poor way to detect mural ulceration anyway, since a thrombus-filled ulcer will not show as a defect on the angiogram, while an empty ulcer crater shows nicely but contains no thrombus!

Our workup is changed by the knowledge that a patient, symptomatic or asymptomatic, has a lesion with a 1.5 mm rather than a 2.7 mm residual lumen diameter. The 1.5 mm lesion is a potential threat even if asymptomatic while the 2.7 mm lesion is hardly ever a source of symptoms. A patient with neurological symptoms lateralized to the side of such a modest carotid stenosis should have a search for more proximal sources of embolus.

Oculoplethysmography is a relatively insensitive indirect method of carotid diagnosis. Spectral bruit analysis, by contrast, has improved markedly since the 5 and 8 year old studies cited by the author. In a more recent direct comparison with pathologic specimens, it was accurate to within 0.5 mm in 94% of 33 patients in whom analyzable bruits were present. In the same study, selective carotid angiography was accurate to within 0.5 mm in 35 of 37 studies in which analyzable films were obtained. Thus, angiograms were not significantly more accurate than spectral bruit analysis in a blinded comparison, a point of which Dr. Berguer seems to be unaware. Furthermore, of the 39 patients in that study, 6 had spectral bruit analyses which could not be analyzed, and 2 more had stenoses which were ± 1 mm different from the estimated sources of embolus.

Two clinical points deserve comment. First, what on earth is a nonlateralizing TIA? Second, the 98% stenosis is hardly “likely to be silent and thus go undetected.” It usually makes a spectacular, high-pitched, almost continuous bruit. Furthermore, duplex doppler ultrasound is highly accurate in detecting lesions in such cases and one seldom uses a single noninvasive method to evaluate a patient.

In summary, carotid atherosclerosis need not be operated on simply because it is present. Knowledge of its natural history is important because it teaches us that most patients with carotid atherosclerosis have no symptoms from it, and that patients with neurological symptoms and carotid disease require careful clinical evaluation of their entire vascular system. Available carotid noninvasive techniques are highly accurate and allow many patients and their physicians to avoid unnecessary angiography, with its attendant risk and expense. Conversely, patients with equivocal symptoms may have accurate diagnosis made and severe.
Further Comment

Another View of Carotid Artery Noninvasive Testing

WILLIAM GEE, M.D.

To the Editor:

Elsewhere in this issue, Dr. Ramon Berguer indicates that carotid artery noninvasive testing is being overused. One theme of his presentation is that hemispheric or monocular transient ischemic attacks are frequently caused by ulcerating nonstenosing lesions, and that these lesions are easily missed by the various noninvasive techniques. To the contrary, Busuttil and his colleagues reported that the incidence of transient ischemic attack and stroke is low, in patients with carotid lesions of no hemodynamic consequence. They investigated the OPG-Gee to define the presence or absence of unilateral or bilateral carotid lesions of pressure-significance, as defined by Berguer and Hwang. An overall accuracy of 98% has recently been published for this technique.

Another theme in the remarks by Dr. Berguer is that CNS symptoms which are neither hemispheric nor monocular have multiple causes other than a carotid etiology. However, if the symptoms are due to a silent severe carotid stenosis, thrombotic occlusion and stroke can occur, while the patient is being evaluated for a suspected noncarotid cause. In this group of patients the OPG-Gee is used as a preliminary screening device, much as is the ECG in patients with chest pain.

As noted by Dr. Berguer, the literature abounds with papers on a variety of noninvasive tests for the evaluation of carotid disease. The OPG-Gee was designed with a single purpose, the simultaneous assessment of the quantitative physiology of the entirety of both carotid arterial hemisystems, including variations induced by cardiac function. Although Dr. Berguer does distinguish the various forms of ocular plethysmography somewhat, so other OPG duplicates the function of the OPG-Gee, nor does any other form of noninvasive carotid evaluation. It was designed for simplicity of application, rapidity of testing (less than one minute) and ease of interpretation.

Standard arteriography, intravenous digital subtraction angiography and the ultrasonic techniques of imaging and scanning are all directed at the anatomic definition of the two carotid arterial hemisystems. The ultrasonic techniques are widely applied in the clinical setting in the United States, yet Dr. Berguer indicates that they are best suited to the research sphere. It may be the ultrasonic techniques that Dr. Berguer feels are overused clinically. This does not detract from the proponents of these instruments, who have developed an enviable expertise, especially in the anatomic definition of minimal to moderate carotid bifurcation atherosclerosis. However, as these individuals acknowledge, technical adequacy requires six months of intensive effort, and technical proficiency requires one to two years. Proximal and distal disease of the carotid arterial hemisystems is poorly assessed by these techniques. It is unfortunate that many laboratories which have attempted to duplicate these techniques have had unacceptable results, which has precipitated statements that intravenous digital subtraction angiography (IDSA) will replace the ultrasonic techniques. As noted by Dr. Berguer, the ultrasonic techniques will retain a definite area of superiority. They allow the frequent examination of the carotid bifurcation, without the risks of dye injection or cumulative radiation. They are ideal for the study of the natural history of the progression of carotid bifurcation atherosclerosis.

Dr. Berguer concludes by observing that the initial enthusiastic claims for intravenous digital subtraction angiography (IDSA) are somewhat premature, but that improvements are both possible and expected. Strother and Crummy, early proponents of IDSA, have also urged caution in this regard. In addition, they noted "the fact that clinical manifestations of thrombo-embolic stroke are, for the most part, related more to physiological changes than to disturbed anatomy . . . .", an observation with which I agree (underlining is mine).

In summary, the following is a recommended algorithm. In the asymptomatic patient with an increased likelihood of carotid atherosclerosis (coronary or peripheral atherosclerosis, carotid bruit, neck radiation) an OPG-Gee study is done at six-month intervals. If the initial or any subsequent test is positive, standard arteriography is recommended. In the symptomatic patient (global, hemispheric, monocular, binocular) with a positive OPG-Gee, standard arteriography is recommended. In the patient with hemispheric or monocular symptoms and a negative OPG-Gee, standard arteriography is recommended. In the group of patients with global or vertebral basilar symptoms and a negative OPG-Gee, our experience has shown the highest incidence of cardiac dysrhythmia. One of the four channels of the OPG-Gee record carries the simultaneous ECG. It is amazing to see the profound alteration in ocularvascular dynamics associated with what superficially appear to be minor cardiac dysrhythmia. Some of these problems are
treated with the implantation of ventricular demand pacemakers. Our experience has documented that in somewhat over one-quarter of patients so treated, little improvement is noted. The lack of improvement is directly attributable to pacemaker effect, and conversion to atrioventricular sequential pacing corrects the deficiency.

Standard arteriography in our community hospital has a mortality of less than 0.1% and a morbidity of less than 1.0%. The unsurpassed anatomic definition afforded by this technique, coupled with the physiologic assessment provided by the OPG-Gee, remains our primary method, for the present. A combination of the OPG-Gee and IDSA, both as outpatient procedures, may reduce the inpatient requirement for standard arteriography, in the future.

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Addendum
The therapeutic implications of the previous comments are considerable. Many papers related to the anatomic definition of carotid atherosclerosis without stenosis of hemodynamic consequence strongly endorse endarterectomy, if an "ulcer" is demonstrated. Many other papers document the frequent disagreement between the preoperative anatomic assessment of carotid atherosclerosis and the actual appearance at operation. However, two of the papers previously cited indicate that the incidence of transient ischemic attack and stroke, secondary to embolus or hypoperfusion, is low in patients without stenoses of hemodynamic consequence.

Although carotid endarterectomy has reduced the incidence of transient ischemic attack and stroke in patients managed by competent teams, antiplatelet aggregating regimens have also been successful in reducing the incidence of transient ischemic attack. What is less clear is the effect of these latter regimens on the incidence of stroke. It has been argued that they may actually hasten the development of atherosclerotic stenoses or mask the natural progression of them until critical degree of stenosis with its associated thrombosis results in stroke.

At this institution, in serial OPG-Gee evaluations of patients with asymptomatic carotid bruits, progression from a lesion of no hemodynamic consequence to total occlusion, the latter as demonstrated by arteriography, has been demonstrated to occur in periods of less than one year. However, progression of this magnitude has never been observed in a period of less than six months. Current policy is serial evaluation at six-month intervals, as long as the OPG remains negative. The patient immediately becomes a candidate for operation if the test becomes positive. This same plan of action could be applied to patients treated by physicians who prefer a drug regimen for initial management of transient ischemic attacks, in patients with negative OPG tests. If symptoms recur, or if the test becomes positive, arteriography and operation are indicated.
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